AMENDMENTS TO THE CLAIMS:

1-15 (**Canceled**)

16. (**Currently Amended**) A method of inhibiting a binding event between a target protein (T) and a binding protein (P), comprising:

administering to a cell **in vitro** an effective amount of a non-naturally occurring bifunctional inhibitor molecule (I) of less than 5000 daltons consisting essentially of:

(a) a target protein ligand that specifically binds to a target protein (T); and

(b) a blocking protein ligand that specifically binds to a blocking protein (B),

wherein said target protein ligand and said blocking protein ligand are covalently bonded to each other, optionally through a linking group;

in order to non-covalently bind the target protein (T) and the blocking protein (B) to produce a tripartite complex (T-I-B) that prevents access of the binding protein (P) to the target protein (T).

17. (**Original**) The method according to Claim 16, wherein said bifunctional inhibitor molecule comprises a linking group.

18. (**Previously Presented**) The method according to Claim 16, wherein said bifunctional inhibitor molecule (I) binds to a site of said target protein that is also bound by said binding protein (P).

19. (**Previously Presented**) The method according to Claim 16, wherein said bifunctional inhibitor molecule (I) binds to a site of said target protein (T) that is not bound by said binding protein (P).

20. (**Original**) The method according to Claim 16, wherein said tripartite complex is produced intracellularly.

21. (**Original**) The method according to Claim 16, wherein said tripartite complex is produced extracellularly.

22. (**Previously Presented**) The method according to Claim 16, wherein said blocking protein (B) is endogenous to said cells.

23. (**Previously Presented**) The method according to Claim 22, wherein said blocking protein (B) is selected from the group consisting of: peptidyl-prolyl isomerases, Hsp90 (Heat shock protein 90), steroid hormone receptors, cytoskeletal proteins, albumin and vitamin receptors.

24. (**Previously Presented**) The method according to Claim 16, wherein said bifunctional inhibitor molecule (I) is administered as a pharmaceutical preparation.

25.-48. (**Canceled**)

49. (**Currently Amended**) A method of inhibiting a binding event between a target protein (T) and a binding protein (P), comprising:

administering to a cell **in vitro** an effective amount of a non-naturally occurring bifunctional inhibitor molecule (I) of less than 5000 daltons consisting essentially of:

(a) a target protein ligand that specifically binds to a target protein (T) with a binding affinity of at least about 10-4 M; and

(b) a blocking protein ligand that specifically binds to a blocking protein (B), wherein said blocking protein ligand is a peptidyl-prolyl isomerase ligand,

wherein said target protein ligand and said blocking protein ligand are covalently bonded to each other, optionally through a linking group;

in order to non-covalently bind the target protein (T) and the blocking protein (B) to produce a tripartite complex (T-I-B) that prevents access of the binding protein (P) to the target protein (T).

50. (**Previously Presented**) The method according to Claim 49, wherein said bifunctional inhibitor molecule comprises a linking group.

51. (**Previously Presented**) The method according to Claim 49, wherein said bifunctional inhibitor molecule (I) binds to a site of said target protein that is also bound by said binding protein (P).

52. (**Previously Presented**) The method according to Claim 49, wherein said bifunctional inhibitor molecule (I) binds to a site of said target protein (T) that is not bound by said binding protein (P).

53. (**Previously Presented**) The method according to Claim 49, wherein said tripartite complex is produced intracellularly.

54. (**Previously Presented**) The method according to Claim 49, wherein said blocking protein (B) is endogenous to said cells.

55. (**Previously Presented**) The method according to Claim 49, wherein said bifunctional inhibitor molecule (I) is administered as a pharmaceutical preparation.

56.-64. (**Canceled**)

65. (**Previously Presented**) The method according to Claim 16, wherein said blocking protein ligand is a peptidyl-prolyl isomerase ligand.

66. (**Previously Presented**) The method according to Claim 65, wherein said peptidyl-prolyl isomerase ligand is a ligand for an FKBP or cyclophilin.

67. (**Previously Presented**) The method according to Claim 65, wherein said peptidyl-prolyl isomerase ligand is a ligand for an FKBP.

68. (**Previously Presented**) The method according to Claim 67, wherein said ligand for an FKBP is selected from the group consisting of FK506 and rapamycin.

69. (**Previously Presented**) The method according to Claim 65, wherein said peptidyl-prolyl isomerase ligand is a ligand for a cyclophilin.

70. (**Previously Presented**) The method according to Claim 69, wherein said ligand for a cyclophilin is a cyclosporin.

71. (**Previously Presented**) The method according to Claim 49, wherein said blocking protein ligand is a peptidyl-prolyl isomerase ligand.

72. (**Previously Presented**) The method according to Claim 71, wherein said peptidyl-prolyl isomerase ligand is a ligand for an FKBP or cyclophilin.

73. (**Previously Presented**) The method according to Claim 71, wherein said peptidyl-prolyl isomerase ligand is a ligand for an FKBP.

74. (**Previously Presented**) The method according to Claim 73, wherein said ligand for an FKBP is selected from the group consisting of FK506 and rapamycin.

75. (**Previously Presented**) The method according to Claim 71, wherein said peptidyl-prolyl isomerase ligand is a ligand for a cyclophilin.

76. (**Previously Presented**) The method according to Claim 75, wherein said ligand for a cyclophilin is a cyclosporin.

77.-82. (**Canceled**)